```
     (FILE 'HOME' ENTERED AT 11:28:40 ON 15 JUL 2002)

     FILE 'REGISTRY' ENTERED AT 11:29:37 ON 15 JUL 2002
L1                QUE GCGGCGACTCCGACGCGTCCAGCCCGCGCTCC
L2             30 S L1/SQSN

     FILE 'CAPLUS' ENTERED AT 11:31:34 ON 15 JUL 2002
L3              5 S L2
L4              4 DUP REM L3 (1 DUPLICATE REMOVED)
L5                QUE
TTATACCGCAGGCGGGCGAGCCGCGGGCGCTCGCT|CCGAGAGCCCTGCGGGGCCCGCC
                  S L5/SQSN

     FILE 'REGISTRY' ENTERED AT 11:33:38 ON 15 JUL 2002
L6             13 S L5/SQSN

     FILE 'CAPLUS' ENTERED AT 11:33:56 ON 15 JUL 2002
L7              5 S L6

     FILE 'CAPLUS' ENTERED AT 11:34:01 ON 15 JUL 2002
L8              5 S L7
L9              1 S L8 AND METHYLA?
L10            18 S MYOD AND METHYLA?
L11             1 S MYOD AND MYF-3
L12             0 S MYF3 AND METHYLA
L13             1 S MYF3 AND METHYLA?
L14             6 S MYF 3 AND METHYLA?

     FILE 'STNGUIDE' ENTERED AT 11:37:58 ON 15 JUL 2002

     FILE 'REGISTRY' ENTERED AT 11:40:17 ON 15 JUL 2002
L15               QUE
CTCCAGCGAAGGCCTCGCGGCCTCCGAGCCTTATAAG|GGGGACGCGGGCCGCGCGTAC
L16             2 S L15/SQSN

     FILE 'CAPLUS' ENTERED AT 11:41:25 ON 15 JUL 2002
L17             1 S L16
L18            30 S GSTPI OR GLUTATIONE-S-TRANSFERASE?
L19             6 S L18 AND METHYLA?

     FILE 'MEDLINE, BIOSIS' ENTERED AT 11:43:19 ON 15 JUL 2002
L20             5 S L19
L21             4 DUP REM L20 (1 DUPLICATE REMOVED)
L22           153 S GSTPI OR GLUTATIONE (7A) TRANSFERASE?
L23             0 S L22 AND METHYLA
L24             5 S L22 AND METHYLA?
L25             1 S L24 NOT L21

=>
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L Number	Hits	Search Text	DB	Time stamp
3	524	(hybridi$7) same (detect$) same (melt$7 or Tm) same (different$7 or higher or lower)	USPAT	2002/07/15 08:46
4	166	(hybridi$7) same (detect$7) same (melt$7 or Tm) same (different$7 or higher or lower) same (mismatch$3)	USPAT	2002/07/15 08:56
5	3414	different$5 near5 hybridiz$	USPAT	2002/07/15 08:57
6	1985	different$5 near2 hybridiz$	USPAT	2002/07/15 08:57
7	537	differential adj1 hybridization	USPAT	2002/07/15 08:57
8	0	differential adj1 hybridization same (FRET)	USPAT	2002/07/15 08:57
9	0	differential adj1 hybridization same (quencher$ or fluorophore)	USPAT	2002/07/15 08:58
10	0	(differential adj1 hybridization) same (quencher$ or fluorophore)	USPAT	2002/07/15 08:58
12	0	(differential adj1 hybridization) same (loop$3)	USPAT	2002/07/15 08:58
11	82	(differential adj1 hybridization) same (label$2)	USPAT	2002/07/15 09:00
13	118	(differential adj1 hybridization) same (mismatch$2)	USPAT	2002/07/15 09:07
14	2	(differential adj1 hybridization) same (mismatch$2) same (TM or melt$8)	USPAT	2002/07/15 09:08

L4 ANSWER 1 OF 6 MEDLINE
AN 75184166 MEDLINE
DN 75184166 PubMed ID: 1138935
TI Unusual properties of the DNA from Xanthomonas phage XP-12 in which 5-**methylcytosine** completely replaces cytosine.
AU **Ehrlich M; Ehrlich K;** Mayo J A
SO BIOCHIMICA ET BIOPHYSICA ACTA, **(1975 Jun 16)** 395 (2) 109-19.
Journal code: 0217513. ISSN: 0006-3002.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197509
ED Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19750929
AB Xanthomonas phage XP-12 contains 5-**methylcytosine** completely replacing cytosine. This substitution confers several unusual properties upon XP-12 DNA. The buoyant density of XP-12 DNA in CsCl gradients is 1.710 g/cm-3, 0.16 g/cm-3 lower than that expected for a normal DNA with the same percentage of adenine plus thymine. The melting temperature for XP-12 DNA in 0.012 M Na+ is the highest reported for any naturally occurring DNA, 83.2 degrees C, 6.1 degrees C higher than that of normal DNAs with the same percentage of adenine plus thymine. Unlike the minor amounts of 5-**methylcytosine** found in most plant and animal DNAs, the 5-**methylcytosine** residues of XP-12 derive their **methyl** group from the 3-carbon of serine instead of from the thiomethyl carbon of methionine. .

L13 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2002 ACS
AN 1989:149988 CAPLUS
DN 110:149988
TI Calculated **melting temperature** of **methylated Z-DNA**
AU Hua, X.; Feng, Y.; Prohofsky, E. W.
CS Dep. Phys., Purdue Univ., Lafayette, IN, USA
SO Report (1988), Order No. AD-A193115, 22 pp. Avail.: NTIS
From: Gov. Rep. Announce. Index (U. S.) 1988, 88(18), Abstr. No. 847,050
DT Report
LA English
AB There are 2 approaches to theor. calcn. of DNA melting temp. One is based on a 2 states, quasi-1-dimensional lattice model in which the melting profile and differentiated melting curve could be calcd. as a function of DNA length. Another way is the modified self-consistent effective phonon approxn. (MSPA) in which the dynamic motional behavior of the DNA mol. during the melting process is detailed. The later approach to melting of methylated Z-DNA was used and results were compared to a similar calcn. on unmethylated B-DNA. A calcn. of melting temp. of methylated Z-DNA based on MSPA was presented.

L9 ANSWER 2 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:310675 BIOSIS
DN PREV199900310675
TI Methylation of adenine bases at the N6H2 groups decreases the melting temperature of the DNA duplex independently of the nucleotide sequence.
AU Yamasaki, Tomoko; Yamasaki, Kazuhiko; Suzuki, Masashi (1)
CS (1) AIST-NIBHT CREST Centre of Structural Biology, 1-1 Higashi, Tsukuba, 305-0046 Japan
SO Proceedings of the Japan Academy Series B Physical and Biological Sciences, (Nov., 1998) Vol. 74, No. 9, pp. 210.
ISSN: 0386-2208.
DT Article
LA English
SL English
AB The effects of methylating adenine bases at the N6H2 groups on the thermal denaturation of the oligomer DNA duplexes were analyzed. Methylation of four adenine bases in a decamer DNA duplex decreased the melting temperature, **Tm**, by 9.4 degrees. **Methylation** of two adenine bases each in various dodecamer DNA duplexes decreased Tm by approximately 4 degrees. These effects correspond to destabilization of the duplexes by 0.6+-0.1 Kcal/mol per each methylation, and were essentially indepent of the length, the nucleotide sequence, and the number and positions of the methylated adenine bases incorporated. A possible biological function for methylation of adenine bases in destabilizing genomic DNA duplexes for the initiation of the DNA replication is discussed.

L9 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2002 ACS
AN 1969:418769 CAPLUS
DN 71:18769
TI Effects of methylation on the behavior of deoxyribonucleic acid
AU Leng, Marc; Rosilio, Charles; Boudet, J.
CS Centre Biophys. Mol., Orleans, Fr.
SO Biochim. Biophys. Acta (1969), 174(2), 574-84
CODEN: BBACAQ
DT Journal
LA French
AB Methylation of native DNA by dimethyl sulfate at pH 6.6 in 1M NaCl gives a product in which about 40% of the guanine residues are methylated at N-7. There is no degradation and no loss of secondary structure of DNA as shown by measurements of light scattering, intrinsic viscosity (.eta.), sedimentation and melting curves. In comparison with native DNA, the melting temp., Tm, is decreased (14.5.degree. for Micrococcus lysodeikticus with 40% methylated guanine residues). The variation of .eta. and Tm vs. pH differs notably from that of untreated DNA, giving some information on the process of protonation. Spermine and spermidine decrease the **Tm** of **methylated** DNA, while under the same conditions the Tm of DNA is increased. The different effect of spermine on unmethylated and methylated DNA is also shown by circular dichroism. The synthesis of RNA by RNA polymerase using methylated DNA as matrix is very much reduced, despite the lower stability of this product.

L14 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:274067 CAPLUS
DOCUMENT NUMBER: 131:114389
TITLE: DNA **methylation** and developmental genes in lymphomagenesis-more questions than answers?
AUTHOR(S): Kay, Peter H.; Spagnolo, Dominic V.; Taylor, Jeremy; Ziman, Melanie
CORPORATE SOURCE: Molecular Pathology Laboratory, Department of Pathology, University of Western Australia, Western Australia, Australia
SOURCE: Leukemia & Lymphoma (1997), 24(3/4), 211-220
CODEN: LELYEA; ISSN: 1042-8194
PUBLISHER: Harwood Academic Publishers
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review, with 58 refs. There is now considerable evidence suggesting that alterations in the DNA **methylating** machinery play an important role in tumorigenesis and tumor progression. For example, focal hypermethylation and generalized genomic demethylation are features of many different types of neoplasms. It is thought that tumorigenesis and tumor progression may be caused by hypermethylation-induced mutational events and silencing of genes which control cellular proliferation and/or demethylation-induced reactivation of genes which may only be required during embryol. development. Consequently, the authors have begun to investigate the role of DNA **methylation** and developmental genes in malignant lymphoproliferative diseases. Previously, in all cases of non-Hodgkin's lymphoma and leukemia studied, the myogenic developmental gene **Myf-3** is abnormally hypermethylated. In this review, the authors discuss the possible significance of these findings since in vitro studies suggest that **Myf-3** may play an important role in control of the cell cycle and therefore lymphomagenesis. In vitro and in vivo evidence suggests that PAX genes may also have oncogenic potential. The PAX family of developmental genes are involved in cellular differentiation, proliferation and cell migration. Expression of PAX3 in particular is assocd. with cellular mobility. Previous studies have indicated that alternate regional expression of PAX genes may be controlled by DNA **methylation**. Therefore, the authors have proposed that abnormal **methylation** profiles of PAX3 may be assocd. with neoplastic transformation and/or metastatic potential. Results thus far reveal that the paired box of PAX3 is abnormally hypermethylated and the homeobox abnormally hypomethylated in lymphomas and leukemias. These new findings are consistent with the authors' postulate and support the idea that inappropriate **methylation** induced activation or inactivation of developmental genes such as **Myf-3** and PAX3 play an important role in lymphomagenesis and disease progression and that inspection of the **methylation** status of other developmental genes is warranted.
REFERENCE COUNT: 58 THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:49687 CAPLUS
DOCUMENT NUMBER: 128:113532

TITLE: Evidence that DNA **methylation** imbalance is not involved in the development of malignant mesothelioma

AUTHOR(S): Bagwe, Aparna N.; Kay, Peter H.; Spagnolo, Dominic V.
CORPORATE SOURCE: Molecular Pathology Laboratory, Department of Pathology, The University of Western Australia, Nedlands, 6907, Australia

SOURCE: Anticancer Research (1997), 17(5A), 3341-3343
CODEN: ANTRD4; ISSN: 0250-7005
PUBLISHER: Anticancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Methylation** dysregulation has been a consistent finding in various malignancies, particularly those where the pathogenetic mechanisms are unclear. To test the hypothesis that **methylation** imbalance may not be a feature of cancers where the etiol. agent or process is known, the authors studied the **methylation** status of the myogenic genes **Myf-3** and Myf-4 by Southern blotting in malignant mesothelioma, a cancer strongly assocd. with asbestos exposure. DNA samples obtained from controls and mesothelioma patients did not exhibit hypermethylation of **Myf-3** and hypomethylation of Myf-4, as noted in malignant lymphomas. The **methylation** status of **Myf-3** and Myf-4 in malignant mesothelioma was similar to that of non-malignant cells indicating that dysregulation of the DNA **methylating** machinery may not be involved in mesothelioma development. The present findings do not support the view that **methylation** imbalance is a consequence of neoplastic transformation, but indicate that it may be one of the early mol. events involved in the genesis of some cancers.